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Effects of He-O_2 breathing during experimental decompression sickness following air dives

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Catron PW, Thomas LB, Flynn ET Jr, McDermott JJ, Holt MA. Effects of He-O_2 breathing during experimental decompression sickness following air dives. Undersea Biomed Res 1987; 14(2):101–111.—The effects of ventilation with He-O_2 during decompression sickness (DCS) and venous air embolism were studied. Fifteen anesthetized dogs were mechanically ventilated and subjected to repeated air dives until pulmonary artery pressure at least doubled within 10 min postdive. At 30 min postdive, ventilation was either continued with air (controls, $n = 7$) or changed to He-O_2 ($n = 8$) for an additional 90 min. All animals developed pulmonary hypertension, systemic hypotension, hemoconcentration, hypoxemia, hypercarbia, and pulmonary edema. Breathing air or He-O_2 postdive did not alter these responses, but He-O_2 breathing produced an 11% increase in pulmonary vascular resistance (PVR). In 3 other anesthetized dogs that were not subjected to dives, ventilation was changed to He-O_2 at various times during an intravenous infusion of air; He-O_2 breathing caused a 22% increase in PVR. We conclude that breathing He-O_2 during DCS resulting from an air dive can intensify pulmonary vascular obstruction.

diving
decompression sickness
venous air embolism
helium-oxygen
chokes
pulmonary edema
animals

Decompression sickness (DCS) is a well-established hazard of air diving. The use of He-O_2 mixtures for treatment of DCS resulting from an air dive has been previously advocated, but the proposed benefits are supported only by anecdotal evidence (1–5). Due to the different diffusivities of helium and nitrogen, breathing He-O_2 in air DCS could theoretically pose a significant hazard. Transient bubble growth resulting from a more rapid diffusion of helium into the bubble than nitrogen out of it could materially worsen symptoms.

The central location of the lung within the circulation makes it a major target organ in DCS. Bubble embolization of the lung during DCS regularly results in pulmonary hypertension and impaired gas exchange (6–8). These responses might be expected to increase if breathing He-O_2 caused growth of nitrogen-filled bubbles within the

pulmonary circulation. This hypothesis is supported by the experimental observation that ventilation of animals with He-O_2 during venous air embolism worsens pulmonary hypertension and hypoxemia (9). To test this hypothesis, we studied the effects of He-O_2 breathing in an animal model of DCS.

MATERIALS AND METHODS

Nineteen experiments were completed using male mongrel dogs. The dogs were divided into 3 groups, an air control group (n = 7), an He-O_2 group (n = 8), and an air embolism group (n = 3). Mean weights (± SD) of each group were: air controls 12.6 ± 0.5 kg; He-O_2 16.2 ± 2.6 kg; and air embolism group 12.3 ± 0.8 kg.

Fasted animals were anesthetized with a loading dose of sodium pentobarbital (30 mg/kg, i.v.), endotracheally intubated, and placed on a mechanical ventilator breathing air. Anesthesia was maintained by a continuous intravenous infusion of sodium pentobarbital (0.068 mg · kg^{-1} · min^{-1}), supplemented with intravenous boluses of 50 mg as needed to suppress corneal reflex and spontaneous ventilation.

A pressure-cycled ventilator (Bird Products, Palm Springs, CA) was used during surgical preparation. At all other times a constant-volume ventilator (Penlon Ltd, Abingdon, England) was used. Before the predive data collection the ventilator was adjusted to provide physiologic values of arterial pH, oxygen partial pressure (P_{O_2}), and carbon dioxide partial pressure (P_{CO_2}). After this adjustment no further change in ventilator volume or rate was made during the experiment. The oxygen content of the He-O_2 mixture used in the He-O_2 group was measured using a Beckman OM-11 oxygen analyzer (Beckman Corp., Schiller Park, IL). The inspired He-O_2 mixtures always contained between 20.5 and 20.9% oxygen.

A 7-French Swan-Ganz catheter was advanced through a femoral vein into the pulmonary artery to measure pulmonary artery pressure and cardiac output (CO). For the measurement of systemic arterial pressure, a 1.77-mm i.d., fluid-filled catheter was advanced through one femoral artery into the midthoracic aorta. To measure left ventricular end-diastolic pressure (LVEDP), a 0.96-mm i.d., fluid-filled catheter was placed in the other femoral artery and advanced retrograde across the aortic valve into the left ventricle. Vascular pressures were measured using Gould P23 pressure transducers and Gould pressure processors (Gould, Cleveland, OH). All vascular pressures were referenced to midchest level. CO was measured using a thermodilution cardiac-output computer (Edwards Laboratories, Santa Ana, CA). Mean pulmonary artery pressure (PAP), CO, and LVEDP were used to calculate pulmonary vascular resistance (PVR). All animals were instrumented with electrocardiographic leads, a rectal temperature probe, and a urinary bladder catheter.

Baseline predive measurements were made of core body temperature, CO, hematocrit (Hct), arterial P_{O_2}, P_{CO_2}, and pH, and vascular pressures. In animals of the He-O_2 group, predive measurements were made of all variables during ventilation with air and with He-O_2. After baseline measurements, the vascular catheters were filled with a volume of heparinized saline (5 U/ml) equivalent to catheter volume, and animals were subjected to a simulated air dive to 10 ATA in an animal recompression chamber (Hahn and Clay, Houston, TX). For all dives the descent rate to 10 ATA was 3.0 ATA/min (3-min descent). The ascent rate from 10 ATA was 1.8 ATA/min (5-min ascent). For the first dive the time at 10 ATA was always 14 min. After the

animals had returned to the normal surface pressure of 1 ATA the systolic PAP was monitored for 10 min. If systolic PAP at least doubled during this time, no further dives were made. If lesser pressure increases occurred, repetitive dives each with a 3-min descent, 5 min at 10 ATA, and a 5-min ascent were made until systolic PAP at least doubled during the postdive observation period of 10 min.

Postdive measurements of physiologic variables were made at 15, 45, 75, and at 105 min after surfacing. These measurements included core temperature, Hct, arterial P_{O_2}, P_{CO_2}, and pH, CO, and vascular pressures. Thirty minutes after surfacing, the inspired gas was either changed to He-O_2 (He-O_2 group) or mechanical ventilation was continued using air (air control group). Vascular pressures were recorded continuously for 10 min before and after switching to the test gas. CO was measured every 1–2 min for 5 min before and for 10 min after gas switching.

At 120 min postdive, the anesthetized animals were killed with an intravenous bolus of KCl. The lungs were removed, inflated with 10% buffered formalin solution, and prepared for routine histologic examination.

In 3 additional animals (air embolism group), gas switching was studied during venous air embolism at 1 ATA. These animals were anesthetized and instrumented as in the decompression experiments, except that an additional catheter (1.66 mm i.d.) was inserted into the inferior vena cava for infusion of air. In 2 animals, air was infused intravenously for 40 min at a dosage of 0.1 ml · kg^{-1} · min^{-1}, a level that resulted in a stable mean PAP approximately two times the baseline value. At 30 min into the infusion, ventilation was changed to He-O_2 for 10 min while air infusion was continued. In the 3rd animal, air was infused at a rate of 0.35 ml · kg^{-1} · min^{-1} to raise mean PAP to approximately three times baseline values. In this animal ventilation with He-O_2 was begun after 13 min of air embolism and continued for 6.5 min, when it was switched back to air and the air infusion ended. At the end of the air embolism experiments, animals were killed and their lungs removed for histologic examination in the manner described for the decompression experiments.

Statistical analyses were done using either a paired *t* test or an analysis of variance. A paired *t* test was used to compare baseline values of vascular pressures and arterial blood gases during ventilation with air and He-O_2 and to compare mean PAP and PVR before and after gas switching. Two analysis of variance procedures were used. Differences in PVR before and after gas switching were examined using a model that estimated a preswitch value of PVR for each animal and a single percentage increase for the entire group of animals after gas switching. Residual error from an eight-parameter model (no increase postswitch) was compared to that from a nine-parameter model (an equal percentage increase for all animals). Another analysis of variance procedure was used to test the hypothesis that with regard to a given physiologic measurement, control and dived animals were different at one or more times. Physiologic data were fit to a five-parameter model that estimated one mean value for the predive baseline and a mean value at each of the postdive measurement times. The residual error from this model was compared with that from a 10-parameter model in which pre- and postdive means were estimated for both control and dived groups. In both analyses of variance, means were estimated by a least squares method using a FORTRAN version of the iterative parameter estimation program of Bailey and Homer (10). Residual errors were compared using an *F* test, and an alpha level of 0.05 was taken as an indication of statistical significance.

RESULTS

Responses to decompression

The dive profile used in these experiments caused responses that are associated with stressful decompression: pulmonary hypertension, systemic hypotension, decreased CO, systemic acidosis, hypoxemia, hypercarbia, hemoconcentration, and decline in body temperature (6–8). Figures 1 and 2 show that although PAP, mean arterial pressure (MAP), CO, Hct, pH, P_{O_2}, P_{CO_2}, and body temperature all reflected the deleterious effects of the dive, there were no important effects of a switch to He-O_2 (compare *broken* and *solid* lines to the *right* of the *vertical*).

Body temperatures (Fig. 2*d*) of animals of the air group were lower than those of the He-O_2 group, and the differences between the 2 groups were statistically significant ($P < 0.001$). The differences were present predive and remained relatively constant throughout the experiment. The lower temperatures of animals in the air group presumably reflected a more rapid loss of body heat under general anesthesia. This relative inability to maintain thermoregulation may have been the result of a higher ratio of body surface area to mass, because animals in this group weighed less (mean weight 12.6 kg) than those of the He-O_2 group (mean weight 16.2 kg).

Hemodynamic effects

In animals of the He-O_2 group, predive values of mean PAP, PVR, MAP, CO, arterial pH, P_{O_2}, and P_{CO_2} were not significantly different during ventilation with He-O_2 or air. The hemodynamic consequences of changing ventilation from air to He-O_2 during DCS and air embolism are summarized in Fig. 3*a-d*. These figures depict for each animal the average values of mean PAP or PVR for 3 min before and 4 min after the switch to He-O_2. (PVR is expressed in peripheral resistance units [PRU].) One PRU is equal to a pressure drop of 1 mmHg occurring with a flow of 1 ml $\cdot$ s^{-1}. (Normal PVR is approximately 0.2 PRU.) In the decompression experiments, no significant change in mean PAP was seen after the switch to He-O_2 (Fig. 3*a*). PVR, however, rose in these animals from an average of 1.18 PRU to 1.30 PRU after switching to He-O_2, an increase of 11% (Fig. 3*b*). This increase was statistically significant by both paired *t* test and analysis of variance ($P < 0.05$). The increase in PVR with switching was small when compared with the increase produced by the dive itself. After the dive, average PVR rose from 0.17 to 1.176 PRU, an increase of approximately 700%.

By contrast to the decompression studies, during venous air embolism, changing the gas mixture from air to He-O_2 increased mean PAP by an average of 7 mmHg within 4 min of the switch (Fig. 3*c*). PVR rose in these animals by an average of 0.20 PRU after switching to He-O_2, an increase of 24% (Fig. 3*d*).

Increases in PVR and mean PAP after the shift to He-O_2 were much easier to demonstrate in animals with venous air embolism than in those with DCS. Although the sample size was too small for statistical correlations, these differences did not seem to be related to the preexisting level of PVR or CO. In animals with DCS the average preswitch CO was 0.157 liter $\cdot$ min^{-1} $\cdot$ kg^{-1} and the average preswitch PVR was 1.18 PRU. Changing to He-O_2 increased mean PAP by 1 mmHg and PVR by 0.12 PRU (11%). The 1 animal given intravenous air at a rate sufficient to increase baseline mean PAP threefold had preswitch levels of CO of 0.170 liter $\cdot$ min^{-1} $\cdot$ kg^{-1} and PVR

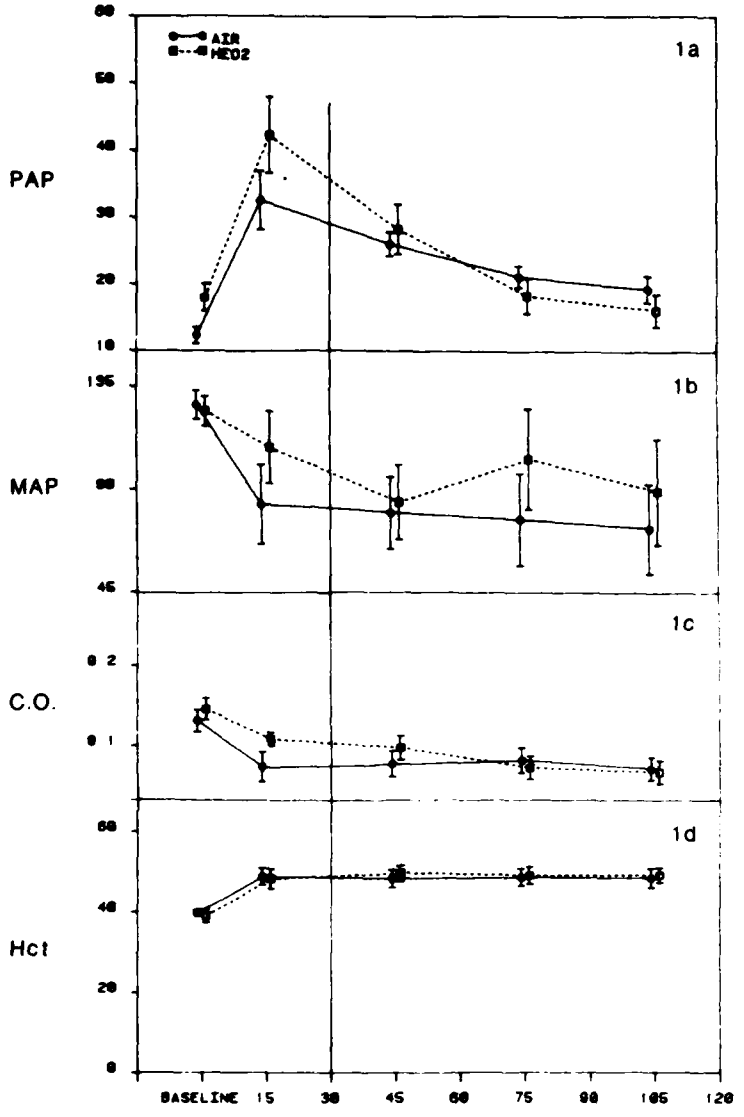

Fig. 1. Mean values of hemodynamic variables during DCS in animals of the air and He-O_2 groups. *a*, Mean pulmonary artery pressure mmHg; *b*, mean systemic arterial pressure mmHg; *c*, cardiac output, liter · min $^{-1}$ · kg $^{-1}$; *d*, hematocrit. In all figures *error bars* denote ± 1 SE and *vertical line* indicates time of switch to He-O_2 breathing.

of 1.26 PRU. Changing to ventilation with He-O_2 in this animal increased mean PAP by 8 mmHg and PVR by 0.29 PRU (22%). Thus, although this animal was comparable to those with DCS in terms of preswitch CO and PVR, switching to He-O_2 caused a substantially greater degree of pulmonary vascular obstruction.

Pathologic changes within the lungs were similar for animals in the air and He-O_2 groups. No animal in either group had normal lungs. The most prominent abnormality was the presence of pulmonary edema. Almost all animals of both groups had cuffs

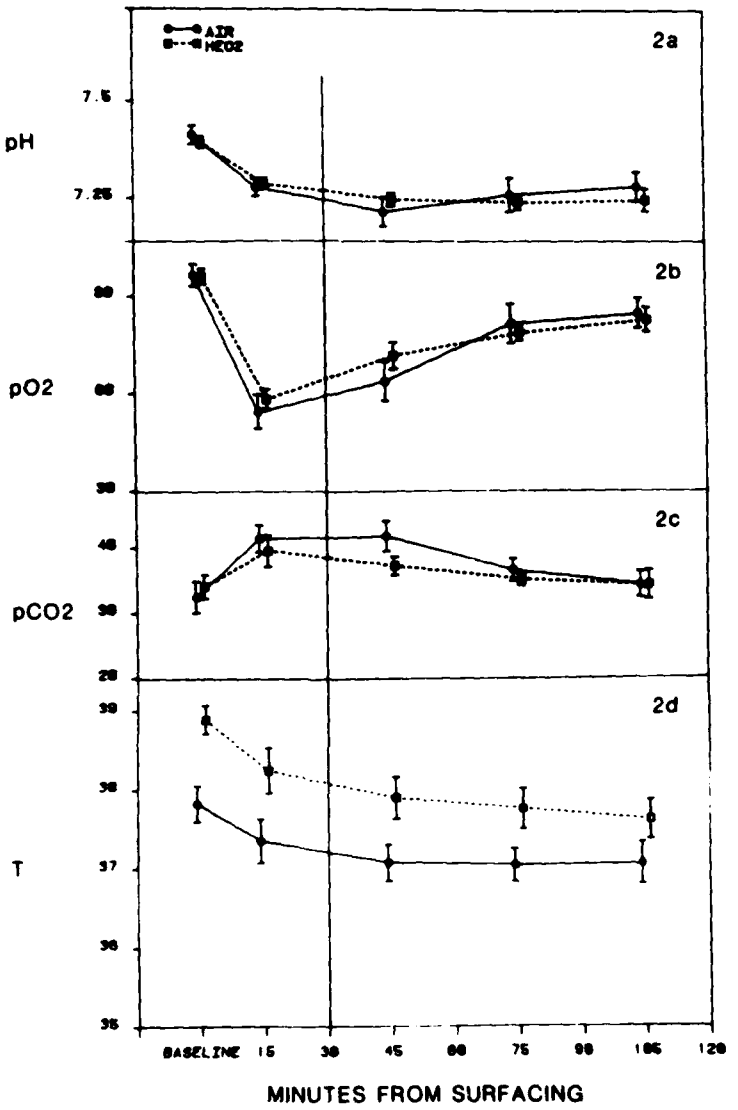

Fig. 2. Mean values of arterial blood gas variables and temperature during DCS in the air and He-O_2 groups. *a*, Arterial pH; *b*, arterial P_{O_2} mmHg; *c*, arterial P_{CO_2} mmHg; *d*, temperature °C. *Error bars* denote ± 1 SE and *vertical line* indicates time of switch to He-O_2 breathing.

of edema fluid encircling pulmonary vessels and bronchi, and some animals in each group had evidence of alveolar filling. There was no apparent difference in severity of the pathologic findings between the two groups.

One animal in the air embolism group had normal lungs. The lungs of the other 2 showed perivascular and peribronchial cuffs of edema fluid. Alveolar filling was not seen in the lungs of any of these animals.

DISCUSSION

Helium-oxygen has been proposed as a suitable breathing mixture for use during recompression therapy of DCS that has resulted from an air dive (1–5). Usually, no

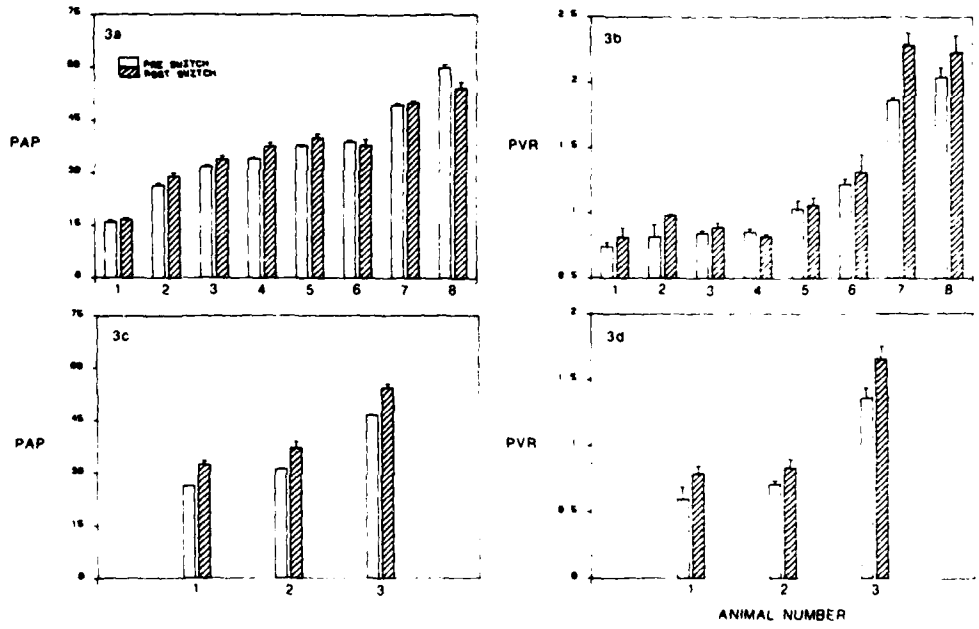

Fig. 3. Mean PAP mmHg (*a*) and PVR (PRU) (*b*) for each animal before and after switching to He-O_2 during DCS. Mean PAP mmHg (*c*) and PVR (PRU) (*d*) for each animal before and after switching to He-O_2 during venous air embolism. *Error bars* denote ± 1 SE.

specific rationale has been offered, and the recommendation implies that He-O_2 is an acceptable substitute for air if its use is operationally convenient. It has also been suggested that He-O_2 might be beneficial in cases of DCS with labored breathing (11) or that it might provide a favorable gradient for the elimination of nitrogen (3). James (12) recommended that He-O_2 be used as a treatment gas for all recompression treatments in lieu of both oxygen and air. He cites lowered breathing resistance and rapid elimination of nitrogen as advantages of He-O_2 gas mixtures.

If a breathing gas has a higher solubility or diffusivity in blood than nitrogen, the risk exists that intravascular bubbles containing nitrogen may temporarily grow through a counterdiffusion mechanism. Such bubble growth might be expected to worsen vascular obstruction.

In the case of bubbles in the pulmonary circulation, the lung may be viewed as an infinite source for inward diffusion of a new gas into intravascular bubbles and an infinite sink for outward diffusion of gas from within bubbles. The effect of gas solubility and diffusivity on changes in bubble size can be appreciated by examination of Fick's law of diffusion expressed in spherical coordinates:

$$dV/dt = \alpha DA\, dP/dr$$

where V is the volume of inert gas, α the solubility and D the diffusivity of the inert gas in blood, P the partial pressure of the inert gas in blood, and r the bubble radius (13).

Assuming that the parameters A and r are the same for the two gases, and P is equal but of opposite sign, the net effect on bubble size of changing breathing gases during DCS will depend on the relative products of solubility and diffusivity of the two gases ($\alpha_1 D_1/\alpha_2 D_2$).

The effect of relative solubility is well illustrated in experiments where nitrous oxide is breathed during the course of venous air embolism. Nitrous oxide is approximately equal to nitrogen in diffusivity (in water at 25°C, $DN_2O = 2.10 \times 10^{-5}$ cm^2/s, $DN_2 = 1.99 \times 10^{-5}$ cm^2/s) (14) but is much more soluble (in blood at 37°C, $\alpha N_2O = 0.455$ ml N_2O/ml blood, $\alpha N_2 = 0.0158$ ml N_2/ml blood) (15). The ratio ($\alpha N_2ODN_2O/\alpha N_2DN_2$) is approximately 30, and this should favor the growth of nitrogen-filled bubbles if the gas surrounding the bubble is abruptly changed to N_2O.

Some experimental observations support this analysis. Changing ventilation of animals from air to a mixture of nitrous oxide and oxygen during venous air embolism has been shown to increase pulmonary artery pressure and arterial P_{CO_2}, and to lower arterial P_{O_2} (9, 16). In addition, the LD_{50} of intravenous air in rabbits anesthetized with halothane, nitrous oxide, and oxygen has been shown to be one-third that of rabbits anesthetized with halothane and oxygen alone; a difference in mortality presumably due to the growth of bubbles in animals anesthetized with nitrous oxide (17). These and similar observations have led to the recommendation that nitrous oxide not be used clinically as an anesthetic agent in cases where a high risk of venous air embolism exists (18–20). Van Liew (21) has shown that rats breathing N_2O-O_2 mixtures after air dives have more severe DCS than controls breathing air.

A similar situation exists for CO_2, which is much more soluble than nitrogen in blood. The ratio $\alpha CO_2 \cdot DCO_2/\alpha N_2 \cdot DN_2$ is approximately 38. Nitrogen-filled bubbles would be expected to grow when surrounded by CO_2. Harvey (22) observed the behavior of nitrogen-filled bubbles as they rose through a water column successively layered with N_2 then CO_2 then N_2 saturated water. Bubbles rapidly enlarged when they encountered the CO_2 layer and shrank when they reencountered a nitrogen saturated layer.

Although He is less soluble than N_2 in blood (at 37°C, $\alpha He = 0.0104$ ml He/ml blood, $\alpha N_2 = 0.0158$ ml N_2/ml blood) (15), it is more diffusible in aqueous media than nitrogen (in water at 25°C, $DHe = 6.2 \times 10^{-5}$ cm^2/s, $DN_2 = 1.99 \times 10^{-5}$ cm^2/s). The ratio $\alpha HeDHe/\alpha N_2DN_2$ is approximately 2. Therefore, nitrogen-filled bubbles would be expected to grow if the gas surrounding the bubbles were abruptly changed from nitrogen to helium. Such an effect has been demonstrated under isobaric conditions with nitrogen-filled bubbles in gelatin (23) and seems to also occur in animals. Sergysels et al. (9) reported that ventilation of animals with He-O_2 during venous air embolism increased mean PAP and lowered arterial P_{O_2}. These changes were thought to be caused by growth of intravascular bubbles within the pulmonary circulation due to rapid inward diffusion of helium.

We found that while He-O_2 breathing did produce a measurable increase in PVR, it did not cause a dramatic worsening of cardiopulmonary function. The most likely explanation for this lies in the severity of the model of DCS used in these experiments. This model corresponds most closely to a "blow-up" of a diver, a situation in which the unplanned ascent of the diver results in a large burden of omitted decompression and severe, usually fatal, DCS. Comparison of our data with that of Powell et al. (24) suggests that the bubble loads in our experiments were several times greater than those required to produce grade IV Doppler bubble signals. When compared to a 700% increase in PVR due to the dive profile used in these experiments, it is unlikely that an additional 11% increase resulting from gas switching would greatly worsen the existing severe impairment of pulmonary function. Although the relative degree of increase in PVR produced by changing ventilation to He-O_2 was small compared

to the dive, it was not small in absolute terms. The average measured increase in PVR produced by switching to He-O_2 was 0.12 PRU, a value which is 70% of the average predive baseline PVR of 0.17 PRU.

Venous gas embolism is not a complete model of DCS. Although the lung's response in the two conditions is similar in many ways, changing to He-O_2 breathing during venous air embolism produces a significantly greater increase in mean PAP and PVR than it does during DCS. This difference in response is not due to differences in severity of the measured pulmonary vascular obstruction before the beginning of He-O_2 ventilation. It may be due to differences in the size of the obstructed vessels in the two conditions. Although we did not measure the size of the obstructing bubbles in these experiments, it is likely that the obstructed vessels were smaller in the decompression experiments. The starting diameter of the air bubbles as they were infused in the air embolism experiments was probably near that of the internal diameter of the infusion catheter (1.66 mm). In a recent study of venous air embolism in which 1-mm diameter air bubbles were infused, bubbles were found in the lung microvasculature with diameters ranging from 100–1000 μm (25). By comparison, many more small bubbles were probably present after decompression. In a previous study in which a resonant bubble detector was used with this DCS model, large numbers of 4-μm-sized bubbles were detected postdive (26). Although it is not possible to predict changes in the size of bubbles as they migrate through the venous system to the lungs, it is likely that the average size of the bubbles obstructing the lung was smaller in the DCS experiments than in the air embolism experiments. If this is true, it may be that many more vessels were completely obstructed during DCS than during air embolism. Growth of bubbles would not be expected to materially worsen vascular obstruction when the bubbles were already totally obstructing small pulmonary vessels. On the other hand, if much of the preswitch pulmonary vascular resistance was due to partial obstruction of larger pulmonary vessels, growth in the size of obstructing bubbles could produce a more prominent increase in measured PVR.

In the decompression experiments, He-O_2 breathing was neither beneficial nor deleterious. However, this study did not measure the effect of He-O_2 breathing during recompression. It is possible that He-O_2 breathing might be beneficial when combined with recompression. The lower density of helium as compared to air could conceivably lower the work of breathing and improve gas mixing in the lung. It has been suggested that such an effect might be helpful in cases of DCS with labored breathing (11). We have previously shown that pulmonary (airways) resistance increases to a variable extent in this model of DCS (6). The increases are relatively small, however, and pulmonary vascular obstruction is probably a much more important cause of the observed derangements of gas exchange than is increased airways resistance. Anticipated benefits of He-O_2 breathing at depth due to lowered gas density would need to be weighed against the potential for worsening pulmonary vascular obstruction.

In most cases the major reason for use of He-O_2 breathing during recompression treatments after air dives is likely to be operational convenience. A decision to use He-O_2 in treatment of air DCS must weigh the risk of worsening vascular obstruction, and therefore outcome of treatment. In this study a definite increase in vascular obstruction occurred when ventilation was changed from air to He-O_2. Although lung function did not deteriorate, a similar increase in vascular obstruction might have very deleterious effects if it occurred in other circulations, such as that of the spinal cord. Although it is difficult to predict the effect of gas switching on bubble growth

in other circulations, one animal study has shown that the efficacy of recompression to 100 fsw for the treatment of spinal cord DCS is less with He-O_2 breathing than air breathing (J. J. W. Sykes, personal communication). In view of these experimental observations, it seems reasonable not to use He-O_2 as a breathing gas for treatment of DCS resulting from air dives until its efficacy and safety have been more completely evaluated by experimental and clinical studies.

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Catron PW, Thomas LB, Flynn ET Jr, McDermott JJ, Holt MA. Effets de la respiration de He-O_2 durant la maladie de décompression expérimentale après des plongées à l'air. Undersea Biomed Res 1987; 14(2): 101–111.—Les effets de la ventilation avec He-O_2 pendant la maladie de décompression (DCS) et l'embolie gazeuse veineuse, ont été étudiés. Quinze chiens anesthésiés furent ventilés mécaniquement et soumis à des plongées répétées à l'air jusqu'à ce que la pression artérielle pulmonaire augmente d'au moins le double en l'espace de 10 min après la plongée. A 30 min après la plongée, la ventilation fut soit continuée avec de l'air (témoin, $n = 7$), ou remplacée avec He-O_2 ($n = 8$) pour une période additionnelle de 90 min. Tous les animaux éprouvèrent une hypertension pulmonaire, hypotension systémique, hémoconcentration, hypoxémie, hypercapnie, et de l'oedème pulmonaire. La respiration d'air ou de He-O_2 après la plongée ne modifia pas ces réponses, mais la respiration de He-O_2 produisit une augmentation de 11% dans la résistance vasculaire pulmonaire (RVP). Chez 3 autres chiens anesthésiés mais qui ne furent pas exposés aux plongées, la ventilation fut changée à l'He-O_2 à des temps variés durant une infusion intraveineuse d'air. La respiration de He-O_2 causa une augmentation de la RVP de 22%. Il est conclu que la respiration de He-O_2 durant la DCS résultant d'une plongée à l'air peut aggraver l'obstruction vasculaire pulmonaire.

plongée
maladie de décompression
embolie gazeuse veineuse
hélium-oxygène
suffocation
oedème pulmonaire
animaux

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